EXHIBIT C

(Excerpts from plaintiffs' provisional patent application No. 61/565,358)

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FILING RECEIPT

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The country code and number of your priority application, to be used for filing abroad under the Paris Convention, is **US 61/565,358**

Projected Publication Date: None, application is not eligible for pre-grant publication

Non-Publication Request: No

Early Publication Request: No
** **SMALL ENTITY** **
Title

METHODS OF GENERATING MESENCHYMAL STROMAL CELLS USING HEMANGIOBLASTS

PROTECTING YOUR INVENTION OUTSIDE THE UNITED STATES

Since the rights granted by a U.S. patent extend only throughout the territory of the United States and have no effect in a foreign country, an inventor who wishes patent protection in another country must apply for a patent in a specific country or in regional patent offices. Applicants may wish to consider the filing of an international application under the Patent Cooperation Treaty (PCT). An international (PCT) application generally has the same effect as a regular national patent application in each PCT-member country. The PCT process **simplifies** the filing of patent applications on the same invention in member countries, but **does not result** in a grant of "an international

[016] FIG. 4 shows the yields of cells positive for MSC surface markers obtained from culturing hESC on gelatin coated plates (first column - no yield), hESC on Matrigel coated plates (second column), and hemangioblasts on Matrigel coated plates (third column).

[017] FIG. 5 depicts the time for MSC surface markers to be acquired using hemangioblasts (top line) and hESC (lower line).

[018] FIG. 6 shows the percentage of cells positive for MSC markers and negative for hematopoiesis and endothelial markers after culturing hESC on Matrigel coated plates (left panel) and hemangioblasts on Matrigel coated plates (right panel).

[019] FIG. 7 depicts the differentiation capabilities of MSCs derived from hemangioblasts differentiated from MA09 hESC to form adipocytes and osteocytes.

[020] FIG. 8 depicts chondrogenic differentiation of MA09 hESC hemangioblast-derived MSCs by mRNA expression of Aggrecan (chondroitin proteoglycan sulfate 1) and Collagen IIa.

[021] FIG. 9 shows the results of a pilot animal study to treat experimental autoimmune encephalomyelitis (EAE) with MSCs derived from hemangioblasts versus vehicle control.

[022] FIG. 10 shows the transient expression of the cell surface marker CD309.

[023] FIG. 11a shows hemangioblast-derived MSCs suppression of T cell proliferation caused by chemical stimulation (PMA/ionomycin).

[024] FIG. 11b shows hemangioblast-derived MSCs suppression of T cell proliferation caused by exposure to dendritic cells.

[025] FIG. 12a shows that hemangioblast-derived MSCs were able to increase the percentage of CD4/CD25 double positive Tregs that are induced in response to IL2 stimulus.

To confirm that the blast-derived MSCs do not contain trace amounts of hESCs, teratoma formation assays will be performed in NOD/SCID mice. 5×10^6 MSCs are injected subcutaneously into the flanks of 4-6 mice. Parent MA09 hESCs will be used as positive controls and the mice will be monitored over the course of 6 weeks to compare teratoma formation in MSC versus hESC-injected mice.

Example 5 - Reduction of EAE Scores by MSCs Derived from Hemangioblasts.

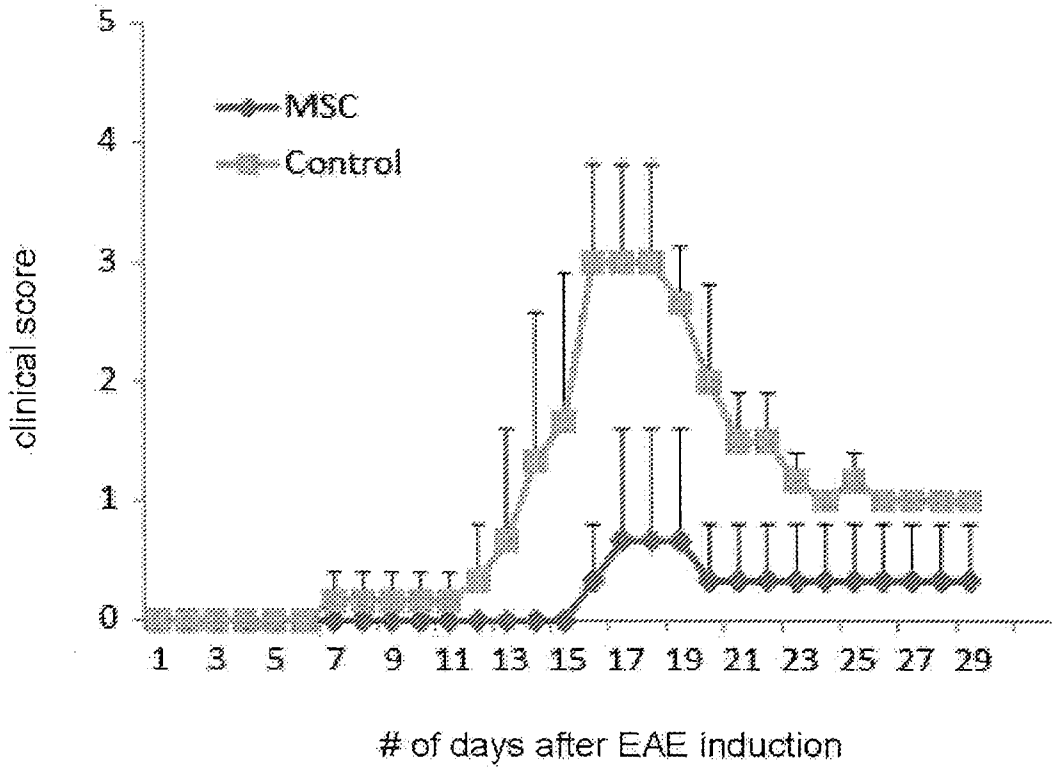
[072] A pilot study to treat experimental autoimmune encephalomyelitis (EAE) on 6-8 weeks of C57BL/6 mice with the hemangioblast-derived hESC-MSCs was conducted. EAE was induced by s.c. injection into the flanks of the mice on day 0 with 100 pL of an emulsion of 50 pg of MOG(35-55) peptide and 250 pg of M. tuberculosis in adjuvant oil (CFA), the mice were also i.p. injected with 500 ng of pertussis toxin. Six days later the mice were i.p. injected with either one million hESC-MSCs in PBS (n = 3) or the vehicle as a control (n = 4). The clinical scores of the animals were recorded for 29 days post the immunization (Fig. 8). A remarkable reduction of the disease scores was observed (Fig. 8).

Example 6 - Confirmation of the Efficacy of hemangioblast-derived hESC-MSCs in EAE Treatment and use of additional animal models of disease

A. Test hESC-MSCs on EAE models in mice confirm their anti-EAE effect.

[073] To confirm the results obtained in Example 5, additional tests are conducted with increased animal numbers, varying cell doses, different administration protocols, and more controls. Clinical score and mortality rate are recorded. The degree of lymphocyte infiltration in the brain and spinal cord of mice will also be assessed. MSC anti-EAE effects is generally thought to involve immunosuppressive activities such as the suppression of Th17 cells and would be expected to reduce the degree of lymphocyte infiltration in the CNS.

Figure 9: One injection of hESC-MSCs can reduce the score of multiple sclerosis symptoms in EAE mouse model



Experimental autoimmune encephalomyelitis (EAE) is delayed in mice treated with hESC-MSCs (n = 3), versus control mice treated with vehicle (n = 4). Clinical score was recorded daily following EAE induction by immunization of the mice with the MOG antigen.